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# CATHETER DUODENOSTOMY: A SAFEGUARD IN GASTRIC RESECTION

## *Report of Eleven Cases*

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THE proper closure of the duodenal stump is a critical step in the safe performance of gastric resections of the Billroth II type. Every surgeon with experience in gastric resections can recall patients in whom leakage from the duodenal stump has resulted in disaster; at best, a stormy convalescence for the patient; at worst, death. Welch[1] deserves the credit for calling attention to the simple maneuver of catheter duodenostomy which can obviate this dangerous complication.

The utilization of a deliberately created duodenal fistula at the time of gastric surgery was reported as early as 1881 by Billroth.[2] Current interest was stimulated by Welch, in 1949, who pointed out the potential usefulness of a catheter placed in the duodenal stump when proximity of pancreatic or common bile ducts might interfere with proper closure; nutritional depletion of the patient with possible impairment of normal healing processes was an additional reason for employing this safeguard. He stated that he had used the procedure in 2 patients. Welch's view was endorsed by Priestley and Butler.[3] They reported 2 cases in which catheter duodenostomy had been performed, and expressed the belief that, when leakage from the duodenal stump was considered a possibility, it was preferable to drain the duodenum directly by catheter rather than the periduodenal area. Ross and Warren[4] mentioned simultaneous catheter duodenostomy and catheter jejunostomy as a safeguard in gastric resection.

It is probable that a safe closure of the duodenum may be performed in nearly every instance if the surgeon will observe three points: (1) take time to mobilize sufficient duodenum; (2) employ pancreatic capsule or fibrous ulcer base as the outer layer of the closure when scar or inflammation makes this desirable; (3) satisfy himself that the gastrojejunostomy is well-placed mechanically, with little likelihood of obstruction. (Back pressure from afferent loop obstruction is probably the principal cause of leakage from a duodenal stump; the rarity with which a perforated ulcer will leak after surgical closure, no matter how flimsy such a closure may be, lends support to this view.)[5] There are, nevertheless, occasional instances in which scarring and inflammation about the duodenum and pancreas will render closure of the duodenal stump difficult and time consuming, and the surgeon will feel uneasy about the safety of the closure. If in addition, the patient is nutritionally depleted and has poor healing propensities, a situation exists where the slightest mal-

functioning of the new gastrojejunostomy may result in leakage. It is in cases of this sort, precisely, where a catheter duodenostomy should be employed; it will save the surgeon time and worry, and may save the patient's life.

In the past 2 years catheter duodenostomy has been performed in 11 instances on one service (S.O.H.) at the Cleveland Clinic. The incidence was, approximately, one in ten gastric resections for all causes. The first person upon whom the procedure was employed (case 1) was an elderly patient undergoing an emergency operation for persistent bleeding from a duodenal ulcer. A formal closure of the duodenal stump (after ligation of the bleeding vessels in the ulcer bed) could have been performed, but the duodenostomy greatly hastened the operation in a poor risk patient. Encouraged by the uneventful convalescence in this patient, we used the procedure upon subsequent patients in whom the duodenal closure appeared to represent an increased hazard. It will be seen from the individual case reports that catheter duodenostomy has been employed with gastric resection for duodenal ulcer, for pyloric and prepyloric ulcer, and for antral cancer with extension into the duodenum. It has been used both with and without a supplementary feeding jejunostomy. (A jejunostomy should undoubtedly be constructed at the time of the catheter duodenostomy if the patient is nutritionally depleted, or if stomal obstruction seems possible on a mechanical basis.)

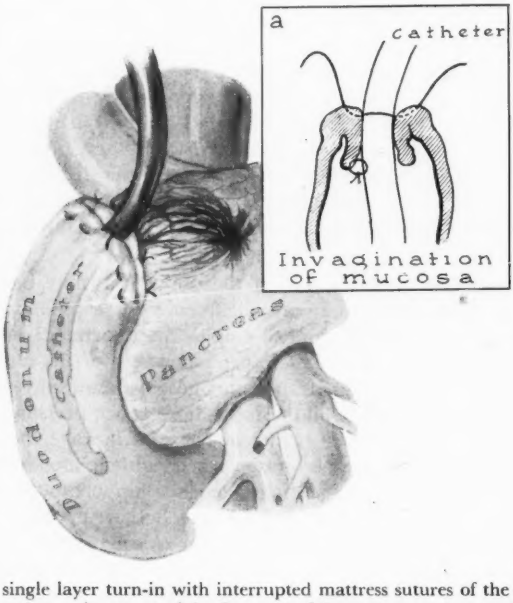


FIG. 1. (a) The single layer turn-in with interrupted mattress sutures of the Halsted type is shown. The insert shows placement of the first part of the mattress suture, and also the suture which anchors catheter to duodenal wall.

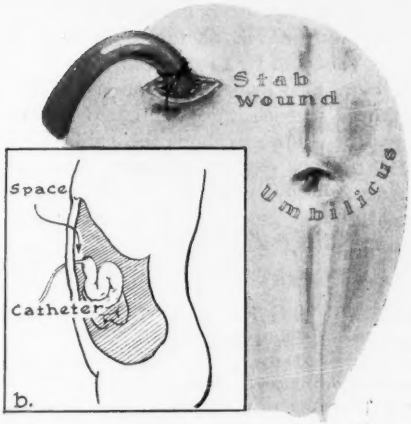


FIG. 1. (b) The usual location of the stab wound is demonstrated. The insert shows the absence of any special walling off as an omental tunnel.

## Technic

The technic employed has been simple. We believe that inversion of the mucosa is the single most important step; failure to obtain a good inversion may result in prolonged drainage after the catheter has been removed (fig. 1a). Mucosal inversion about the catheter may be accomplished by means of a purse-string suture as described by Welch. In the majority of our cases, however, interrupted 000 silk sutures were used in a single layer closure and the catheter led out at any convenient spot, usually the center. A number 14 or 16 French catheter is a convenient size, with additional holes cut at the end. The catheter is introduced for a distance of 4 to 6 inches, and secured to the duodenal wall with a single suture of 000 silk also passing through the wall of the catheter, and tied tightly. (This suture will presumably slough through in 5 or 6 days.) The closure is tested by introducing sterile saline solution through the catheter. The catheter is led out through a stab wound directly over the duodenal stump (fig. 1b). We have not employed a tunnel of omentum, as shown in the illustration in Welch's paper, reasoning that the catheter was analogous to one employed for draining the common duct where such walling off is deemed unnecessary. In several patients there was definite drainage of duodenal contents outside the catheter, either through the same stab wound or by way of an additional drain. This extravasation had no detectable deleterious effect upon the patient.

The postoperative management of the catheter, in our experience, may follow the same principles employed in catheters draining the common bile duct. Gravity drainage may be utilized for the first few days, or even suction drainage if the closure seems insecure. If the drainage is excessive, it may be fed back to the patient through the jejunostomy, if one has been constructed;

such re-feeding will usually not be possible for a day or two, until normal bowel motility has been restored. By the fourth postoperative day, the drainage bottle may usually be raised to bed level. Daily drainage will generally drop to 50 or 100 cc. If there is no leakage around the tube, the catheter may be removed on the sixth or seventh day. Continued drainage of large amounts of duodenal contents, or drainage about the catheter, usually indicates imperfect functioning of the new gastrojejunostomy, and removal of the catheter should be delayed accordingly. Once the catheter is drawn out, the fistula closes rapidly in nearly every case. In one patient the fistula reopened for a few days several months after the operation, and 2 patients developed abscesses in the stab wounds. There were no serious complications related to the catheter duodenostomy.

## Case Reports

**Case 1.** A 58 year old white man came to the Cleveland Clinic in January 1950, complaining of intermittent epigastric distress of 6 years' duration. An x-ray diagnosis of duodenal ulcer had been made in 1944. He was admitted for treatment of a subdeltoid bursitis, but developed massive melena while in the hospital. The bleeding failed to stop despite medical management. After 5 days and transfusions of 3,000 cc. blood, an emergency gastric resection was performed January 30, 1950 for a bleeding, penetrating duodenal ulcer. A catheter duodenostomy was desirable because of fixation of the duodenum to the pancreas by inflammation.

The drainage from the duodenostomy decreased steadily and the catheter was removed on the seventh postoperative day. No further drainage was observed. He was discharged on the ninth postoperative day. The patient was well on follow-up examination in March 1952, 2 years later.

**Case 2.** A 61 year old obese white man came to the Clinic because of ulcer distress of 20 years' duration which had failed to respond to medical treatment. During the preceding 6 months he had experienced 3 episodes of hematemesis and melena.

X-ray examination of the stomach revealed a gastric ulcer which was interpreted as presumably malignant.

On March 3, 1950 the patient had a subtotal gastric resection for a benign prepyloric ulcer. Catheter duodenostomy was performed because of inflammation and induration surrounding the duodenum; obesity contributed to the technical difficulty.

Postoperatively the patient was completely asymptomatic. The duodenostomy drainage steadily decreased and the catheter was removed on the seventh postoperative day. Drainage promptly ceased. The patient was discharged on the thirteenth postoperative day with all wounds healed. In a follow-up by letter in March 1952, 2 years after the operation, the patient was asymptomatic.

**Case 3.** The patient was a 59 year old white woman with a history of 18 years of severe intermittent epigastric distress. In 1946 and twice in 1948 she had operations upon her biliary tract for recurrent jaundice. She continued to experience epigastric distress, chills, fever and jaundice and, in March 1950, was admitted to the Cleveland Clinic Hospital.

X-ray examination of the stomach revealed a duodenal deformity. Operation on March 24 consisted of a transduodenal choledocholithotomy, vagotomy, gastroenterostomy and catheter duodenostomy for a duodenal ulcer and common duct stone. It was felt that the position of the ulcer could have induced the common duct obstruc-

tion. The duodenostomy was performed by introduction of a catheter into the opening already present in the duodenum; this was considered advisable because extensive cicatrization and inflammation rendered an accurate closure impossible.

Postoperatively drainage from the duodenostomy was minimal and the catheter was removed on the tenth day after surgery. No further drainage was observed and the patient was discharged on the fourteenth postoperative day. She was well on follow-up examination on September 26, 1951, 18 months after operation.

**Comment:** This is the only patient in the series in whom the duodenostomy was not an "end" duodenostomy.

**Case 4.** A 58 year old white man came to the Clinic in April 1950 complaining of pain in his epigastrium of about 2 years' duration. The pain had become more severe in the last 2 months and had failed to respond to medical treatment.

X-ray examination of the stomach revealed a large prepyloric ulcer.

On April 10, 1950 a subtotal gastric resection with vagotomy was performed for a chronic peptic ulcer of the pylorus penetrating into the pancreas. Catheter duodenostomy was considered desirable because of extensive induration and inflammation about the duodenum.

Postoperatively the duodenal drainage steadily decreased and the catheter was removed on the eighth day. The fistula rapidly closed and no further drainage was observed at the time of discharge on the tenth postoperative day. He was well on follow-up examination on March 10, 1952, nearly 2 years after operation.

**Case 5.** A 36 year old man came to the Clinic with a long history of intermittent ulcer distress and a draining fistula in the abdomen. In 1939 the patient had an emergency laparotomy for closure of a perforated duodenal ulcer. In 1946 a gastroenterostomy was performed, followed in 2 weeks by incision and drainage of a collection of pancreatic fluid. In 1948 another incision and drainage was performed.

Abdominal examination revealed a tender, movable epigastric mass. X-ray examination of the stomach disclosed a jejunal ulcer and questionable duodenal fistula.

On June 28, 1950 an operation was performed consisting of subtotal gastric resection with vagotomy, resection of the jejunum containing a jejunal ulcer with end-to-end anastomosis, catheter duodenostomy and feeding jejunostomy. The duodenostomy was performed because of induration and cicatrization from the duodenal fistula.

Subsequent duodenal drainage was slight but constant. On the sixth postoperative day the duodenal catheter was removed; the drainage persisted for 4 to 5 days, thereafter clearing completely, and the patient was discharged on the fifteenth postoperative day. Follow-up by letter in September 1951, 15 months after the operation, revealed the patient to be in good health.

**Comment:** Despite the existence of a duodenal fistula prior to operation, closure of the duodenostomy wound was prompt and complete after the catheter was withdrawn.

**Case 6.** The patient was a 64 year old male physician who complained of epigastric burning of 15 years' duration. In 1937 he had an operative repair of a perforated duodenal ulcer. Postoperatively he developed a fistula of duodenal nature which closed spontaneously in a few weeks. In 1941 the patient experienced hematemesis and melena. The patient had a posterior gastroenterostomy in 1949, followed by recurrent epigastric burning in 1950.

X-ray examination of the stomach revealed a gastric ulceration, which was interpreted as presumably malignant.

On August 23, 1950, a subtotal gastric resection utilizing the previous gastroenterostomy was performed for an extensive ulcerated lymphosarcoma of the stomach. Catheter duodenostomy was considered advisable because of the extension of the neoplasm to the pylorus and the presence of additional induration and scar from the old duodenal ulcer.

Postoperatively, drainage from the duodenostomy decreased progressively, although there was some drainage around the catheter as well. The catheter was removed on the fourteenth postoperative day. Intermittent drainage continued for a number of days, but the duodenostomy was closed at the time of discharge from the hospital after completion of a course of x-ray treatments. It reopened for a few days several months after the operation, but closed again spontaneously. At a subsequent follow-up in July 1951, 11 months after the operation, the patient was completely asymptomatic.

**Case 7.** A 50 year old white man came to the Clinic in September 1950 complaining of vague epigastric discomfort of 25 years' duration. He had experienced repeated attacks of this distress associated with vomiting in the previous 3 years. In 1946 he had an operation consisting of a gastroenterostomy followed in 6 months by an enteroanastomosis.

X-ray examination of the stomach revealed hypertrophic gastric rugae, a nonfunctioning gastroenterostomy, pyloric narrowing and a duodenal deformity.

On October 2, 1950 an operation was performed consisting of a gastric resection with resection of the gastroenterostomy, and vagotomy for penetrating duodenal ulcer. Catheter duodenostomy was advisable because of much cicatrization of the entire duodenal wall.

Postoperatively, the patient developed a transient obstruction of the efferent loop of the gastrojejunostomy as evidenced by persistent gastric retention and prolonged drainage from the duodenal catheter. By the twelfth postoperative day the duodenal drainage had steadily decreased and the catheter was removed on the twentieth day. No subsequent drainage was noted and the patient was discharged a week later. He was seen on October 9, 1951, one year after operation; his abdominal wounds were well healed but he had mild steatorrhea.

**Case 8.** The patient was a 53 year old housewife, who came to the Clinic in October 1950. She complained of a gradual onset of upper abdominal, postprandial distress and bloating of 4 years' duration.

Abdominal examination revealed a palpable, movable epigastric mass with some associated tenderness. X-ray examination of the stomach revealed an ulcerating lesion of the distal antrum.

On November 1, 1950 a subtotal gastric resection, catheter duodenostomy, and feeding jejunostomy were performed for scirrhous carcinoma. The catheter duodenostomy was carried out because of wide removal of the duodenum and extensive induration and edema.

Postoperatively, some bile drainage was noted around the duodenal catheter on the fifth day. This continued until the eighth day when the catheter was removed. Minimal duodenal drainage was observed for 3 to 4 days thereafter, but the fistula had closed before the patient was discharged on the fifteenth postoperative day. At follow-up examination in September 1951, 11 months after operation, the patient was asymptomatic.

**Case 9.** The patient was a 55 year old housewife who was admitted to the Clinic on January 26, 1951. She complained of intermittent epigastric distress dating back 4 to 5 years. For 2 to 3 days prior to admission the patient had experienced dizziness, followed by the passage of tarry stools and vomiting of blood. She was in shock on

admission, but improved temporarily with blood transfusions and other suitable measures. When it became evident that active bleeding was still occurring 36 hours after admission, she was taken to the operating room where a subtotal gastric resection, catheter duodenostomy and feeding jejunostomy for bleeding duodenal ulcer were performed. The catheter duodenostomy was considered advisable because of extensive fixation of the duodenum to the head of the pancreas.

Postoperatively the duodenal drainage decreased progressively, and the duodenal catheter was removed on the tenth day. The duodenostomy site became infected and drained slight amounts of semipurulent matter for about 3 months. This ultimately healed and the patient was well in February 1952, one year after the operation.

**Case 10.** The patient was a 47 year old man who complained of epigastric pain of 10 months' duration, and recent vomiting. There had been a 10 pound weight loss.

A movable epigastric mass was noted upon abdominal examination. X-ray examination of the stomach revealed a neoplasm of the stomach.

On July 19, 1951 a subtotal gastric resection, catheter duodenostomy and feeding jejunostomy were performed for carcinoma of the stomach extending into the duodenum. After consultation with the pathologist, a further resection of duodenum had been done to secure a wider margin. The duodenostomy was performed because there was little duodenum left proximal to the ampulla of Vater.

Postoperatively the patient made a satisfactory recovery. Some duodenal drainage was present, but progressively decreased. The catheter was removed on the tenth postoperative day. He was discharged 2 days later with no further drainage from the duodenal fistula. The patient was seen on January 31, 1952, 6 months after operation, at which time he was well.

**Case 11.** A 64 year old white man came to the Clinic complaining of intermittent epigastric distress of 3 years' duration, and weight loss of 45 pounds in less than one year.

X-ray examination of the stomach revealed prepyloric ulceration interpreted as presumably malignant.

On November 9, 1951 a subtotal gastric resection, catheter duodenostomy and feeding jejunostomy were performed for benign pyloric ulcer. The catheter duodenostomy was done because of a severe inflammatory reaction surrounding the duodenal stump.

Postoperatively the patient did well, but slight inflammation was observed around the enterostomy catheters. The duodenal drainage diminished steadily and the duodenal catheter was removed on the seventh postoperative day. No further drainage was noted and the patient was discharged 4 days later. On follow-up examination January 28, 1952, the duodenostomy temporarily opened up via a sinus tract from a wire suture. The suture was removed and, on subsequent examination in February 1952, 3 months after operation, the duodenostomy had completely healed.

## Summary

Catheter duodenostomy is a valuable maneuver in patients where technical difficulties render a tight closure of the duodenal stump difficult or insecure. The technic of the procedure is described, and experiences with 11 patients detailed. There were no deaths and no serious complications in any of these patients.

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# CLINICAL ASPECTS OF ANXIETY STATE AND HYSTERIA

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THE object of this discussion is to offer a simple and practical clarification of the common variety of neuroses and some data in reference to differential diagnosis. Elaborate and exquisite psychodynamic elements in these neuroses are minimized to avoid confusion and to eliminate material which is too theoretical.

Anxiety state is the neurosis most frequently encountered in private practice at the present time. Generally, factors indicative of physical defects are lacking in this type of neurosis. The patient has undoubtedly consulted many doctors who have failed to reassure him or to minimize his persistent symptoms. He describes these in detail and expresses his apprehensions or fears about the cause of his disease. Frequently the attending physician gets the impression that the patient enjoys relating his symptoms but such aeration is probably more relieving than actually productive of pleasure. The attitude of the patient with anxiety symptoms is one of dependence upon the physician, which is secured by emphasis on the psychosomatic tensions. Although the physical symptoms of anxiety state are numerous, they center primarily about the visceral organs. Anxiety neurosis is usually characterized by attacks of dyspnea, tachycardia, sweating and chronic fatigue. The patient accurately and graphically describes "fast heart," "loose bowels," "lump or butterflies in the stomach," "numbness of the hands," or exhaustion. These symptoms frighten the patient and he finds that he is unable to concentrate on his work; he has a constant sense of foreboding, and at times a sense of impending death or insanity. The degree of this anxiety tension fluctuates from time to time and is manifested by crises and remissions in the subjective symptoms. These crises or episodes of acute anxiety state may come during periods of preoccupation with unrelated matters and even during intervals of relaxation. Premonition of impending disaster is continually present, and is decidedly destructive to general efficiency. Because of these crises and total inability to predict them the patient dreads being left alone, clings to his family or friends, and avoids public gatherings.

The source of fatigue in anxiety state is an important consideration because it is too readily attributed to physical factors such as low metabolism, neurocirculatory asthenia and hypotension. Actually it is far more depleting than fatigue based on physical disorder because of its tendency to sudden recurrence and fluctuation. The patient usually relates his history of fatigue in ill-defined terms; generally it is described as inertia, lassitude or loss of animation through-

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out the daily routine. Usual chores are performed with exaggerated effort. Careful interrogation reveals that the fatigue is of a definite selective nature, an important point in the differentiation between organic and functional fatigue. For example, while the patient complains of fatigue he may be pacing the floor and will demonstrate a motor restlessness that denies his basic complaint. Primarily, the fatigue of anxiety state is not relieved by rest or sleep. This is a further differential point in psychogenic and organic fatigue. The patient with an anxiety neurosis feels worse on arising in the morning; this failure to rebuild energy is not always due to insomnia that is invariably associated with anxiety state.

The bases of such neuroses are the hereditary temperament and a predisposition to excessive worry over minor physical irregularities. Acute symptoms frequently arise from forewarnings of insecurity, affront to pride and self esteem, or from sexual irregularities (as postulated by Freud). Specific examples are: (1) loss of social or financial position; (2) domestic conflicts; (3) fear of impotence.

It is easier to recognize an anxiety state if one understands the associated dynamics. The source of conflict or tension in anxiety state is merely suppressed and is, in most cases, due to prolonged situational problems such as marital incompatibility, lack of sexual gratification, and financial insecurity. The patient tries to avoid facing these problems because all previous attempts to solve them have proved unsuccessful. However, this inhibition is never complete or consistent and finally the poorly restrained nervous forces are discharged through psychosomatic channels by way of the autonomic nervous system. One would expect this dissipation of nervous energy to greatly relieve the patient; however these discharges are manifested as the very uncomfortable somatic symptoms previously listed: weakness, fatigue, palpitation, irritable bowel, and vague gastrointestinal complaints. These indications of anxiety may, at times, be so sudden and severe that the patient forgets the significance of the primary problem and fears that the symptoms are a new and added threat of organic origin.

The basis of psychogenic fatigue has been variously explained. Janet's theory of quantitative depletion of nervous energy is implied in the term neurasthenia and was reemphasized by Beard in 1880. Freud, however, established the concept that fatigue is due, not so much to fundamental lack of psychic energy, but to a loss of such energy at unconscious levels where it is drained off by conflicts between opposing instinctual drives. This allegedly leaves but little psychic energy to operate normally at conscious levels.

The next most important type of psychoneurosis is hysteria. The pattern of hysteria is to some degree stereotyped. It develops in people both young and old who have an immature emotional approach to life. These people are often vain, selfish, and coquettish in their reactions. They revel in fantasies, and dramatize themselves. Hysteria can resemble any infirmity which is capable of making a strong and lasting emotional impression on others. The hysterical person unconsciously emulates symptoms of a dramatic nature observed in

others with genuine disease, or conceived by powerful autosuggestion. Therefore, blindness, stupor, epilepsy, and similar disturbances commonly accompany hysteria. The symptoms are either mental or physical.

Although hysterical symptoms develop rather suddenly, careful scrutiny will reveal a definite dissociation of personality which enables the patient to believe in and accept a drastic alteration of his personality capacities. Autosuggestion or powerful external suggestion can permit assumption of a new personality, or of physical characteristics compatible with the newly acquired behavior pattern. This, in the majority of cases, indicates a regression to a lower level of responsibility or efficiency. The acquisition of hysterical symptoms is understandable since the patient is unable to tolerate anxiety or the undue stress imposed by various situations. The new personality is developed as a defense mechanism. By "new personality" we mean a sudden change of apparently organic origin. The patient is unaware that this change or condition has been self-induced. At this point treatment is rather difficult since the patient has slipped to this lower level and consequently has less responsibility and is more comfortably able to cope with situations than he was before his symptoms developed. In this state he may even undergo a regression of his thinking processes, and imply that the pain in his thigh or "stocking anesthesia" needs no explanation, since it actually happened. He is fully content to accept and utilize the infirmity in a juvenile manner. For example, there is the woman who comes into the physician's office complaining of severe abdominal symptoms; she relates a tale of having seen many doctors who have been at a loss to explain satisfactorily the pain. Actually the hysterical person of this type, who has no organic basis for her discomfort, unconsciously wants authoritative confirmation rather than positive relief or a cure. In her case the confirmation of the disease has a psychologic value. A certain type of dissociation is a frequent symptom in hysteria. The patient finds it easy to forget or detach himself from the past, especially if unpleasant situations or problems had existed which were impossible to solve. If this dissociation affects the whole being, it is a fugue or hysterical amnesia. If it affects only a part of the body, then there is usually a focal paralysis or anesthesia, or some similar seemingly organic change. It is interesting to note that when the patient takes flight physically or mentally, as in a fugue, he adopts a personality according to his new concepts, retaining however much of the old behavior pattern. For example, the severely myopic person will rarely discard his glasses.

Physical symptoms are either sensory or motor or both. Hypesthesia and anesthesia take precedence over hyperesthesia in hysterical sensory symptoms. If hyperesthesia or pain is experienced it is, in most cases, mild and rather vague. There is almost always no logical anatomic distribution of the pain.

Hysterical motor symptoms are divided into either irritative or paralytic. Irritative symptoms seem to be more predominant; of them, convulsions, distortion of posture and vomiting seem to be the most common. For example, many of these patients will simulate an epileptic attack with a sudden fall, screaming and severe thrashing of the extremities. However, it is seldom that this type of patient ever injures himself or bites his tongue as is the case in true

epilepsy. Whatever the physical disabilities may be in hysteria, the most important feature is the unnatural indifference which the patient assumes toward his physical handicap.

Mental changes characteristic of hysteria are always alarming. The patient may go into a trancelike state and lie quietly, not even responding to painful stimuli. Such a state is classified as catalepsy. Another mental phenomenon is the previously mentioned fugue or hysterical amnesia. The patient merely blots out periods or situations in his life which are associated with fear or unpleasantness. This type of person is the one who frequently is featured in newspaper headlines, is found wandering in a distant city and appeals to the sympathy and good nature of strangers.

Frequently overlooked is the fact that there are two main types of hysteria, namely conversion and anxiety. As mentioned, conversion hysteria is a state of mental tension that is translated into motor or sensory manifestations which simulate organic disease. However, the patient is not aware of the definite purpose that is being served because the conversion has taken place at unconscious levels. Usually in conversion hysteria the presenting symptom is a somatic disturbance which produces no emotional upheaval.

Anxiety hysteria is a combination of hysteria and anxiety neurosis. The discharge of tension in many instances is through conversion symptoms which represent a definite unconscious wish or purpose. These unconscious wishes are frequently phobic defenses created as a technic by which inner fears and anxieties are projected into outward symbols. To distinguish this from anxiety state one must remember that the latter reflects a continuous sense of disaster —a drifting anxiety, ready to grasp the slightest excuse or concern, fear or worry. However, in anxiety hysteria the process is closer to completion and here the inner fears and anxieties usually have definite symbolization of an outward nature. One must also keep in mind that the discharge of tension is not necessarily through phobic symbolization; it might well be through visceral excitement which represents a definite unconscious wish or purpose.

There are several less common types of hysteria such as anorexia nervosa. This is a hysterical state in which the young woman patient usually refuses to eat, or regurgitates food and, from all outward appearances, is suffering from a progressively debilitating disease. It is almost always precipitated by some problem or emotional disturbance with which the patient cannot cope. Usually an effort to solve the problem, or its complete solution, will result in improvement.

Another form of hysteria is Ganser's syndrome. This is a hysterical reaction upon the part of a prisoner awaiting trial, and it simulates true psychosis for the unconsciously designed purpose of avoiding punishment for a misdeed. The condition is marked by a complete absence of lucid moments. It is interesting to note that hysteria or "hysterics" as known to the layman, is not hysteria at all. Hysterical behavior which is most commonly characterized by alternate spells such as laughing, crying and screaming, is most commonly an indication of nervous instability, which at times of stress affects otherwise normal persons. However, people who are likely to indulge in such outbreaks

are usually immature and somewhat inadequate, but not necessarily neurotic.

In diagnosis and in the differential diagnosis between hysteria and anxiety state (or anxiety neurosis), we must first consider history. With hysteria there is usually a background of sudden onset of some physical or mental manifestation which closely simulates organic disease. If one investigates the history he will find that the presenting and all important symptom is the result of either powerful external suggestion or autosuggestion. These symptoms in hysteria are rather striking such as blindness, deafness, loss of speech, paresthesias and paralysis. Since the hysteriac has a flare for the dramatic, his symptoms will be unusual and outstanding. Frequently one finds that an organic symptom is exaggerated beyond its true value and the patient may feign severe suffering and symptomatology. The hysteriac impresses one with his attitude of indifference toward his rather severe handicap. As mentioned before, the anxiety state is a continual process in which there is a fear of impending doom which is constantly present and accentuated when the patient is resting or when his mind is unoccupied. It is characterized by rather violent somatic visceral symptoms such as dyspnea, tachycardia, sweating, and fatigue. The patient may be intelligent and have excellent insight into the situation (in contrast to the hysteriac); however, he is unable to utilize his intellect to control the attacks. The type of symptom seems to be precipitated by exciting causes based on insecurity or sudden threat to security, loss of prestige before others, or loss of self-respect, position or wealth. This patient is not the immature, short-sighted personality addicted to hysterics.

The main differential point between anxiety neurosis and anxiety hysteria is that, in the latter, the patient develops phobic defenses by means of which inner fears and anxieties are projected into outward symbols. The type of person who develops anxiety hysteria fits into the classification between the short-sighted, immature hysteriac and the introspective, hypochondriacal type who develops anxiety neurosis.

If, after discussing points in differential diagnosis some doubt remains, one might apply the Rorschach test. This test is not only useful in diagnosis but also in evaluating the effects of therapy when before and after records of treatment are kept. Another aid in the diagnosis and differentiation of these psychoneuroses is psychiatric interview under intravenous pentothal or sodium amytal.

## Conclusion

Hysteria and anxiety state should be readily differentiated because they present two separate conditions, and the basic personality of the two types differs in fundamental psychic structure. Hysteria has two major subdivisions, conversion hysteria and anxiety hysteria; and two minor subdivisions, anorexia nervosa, and Ganser's syndrome. Hysterical behavior is not true hysteria, and affects individuals who are not necessarily neurotic. Although the differential diagnosis between hysteria and anxiety state may be made clinically, the Rorschach test and psychiatric interview under sedation are useful adjuncts.

# THE DIFFERENTIATION OF NONINVASIVE AND INVASIVE CARCINOMA ARISING IN INTESTINAL POLYP

## Desmoplastic Tumor Stroma

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and

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ONE of the most perplexing problems confronting the surgical pathologist is the diagnosis of carcinoma in intestinal polyp. It is important to the surgeon that the pathologist not only establish the presence of carcinoma but also the type. Therapy, as outlined in another article,[1] depends upon complete diagnosis, since certain carcinomas arising in polyp may be treated locally without minimizing the chances of cancer cure.

In designating the type of carcinoma arising in polyp, we have used descriptive terms rather than numerical grades (table). Three types are recog-

CARCINOMA ARISING IN POLYP
(Colon)

| Type of Carcinoma | Pathologic Characteristics |
|---|---|
| **Noninvasive** | |
| (a) Carcinoma in situ | Cytologic carcinoma without invasion of basement membrane. |
| (b) Superficial adenocarcinoma | Carcinoma with reduplication of lumina and invasion of basement membrane and lamina propria. |
| **Invasive** | Carcinoma with invasion of *muscularis mucosa* and presence of desmoplastic tumor stroma. |

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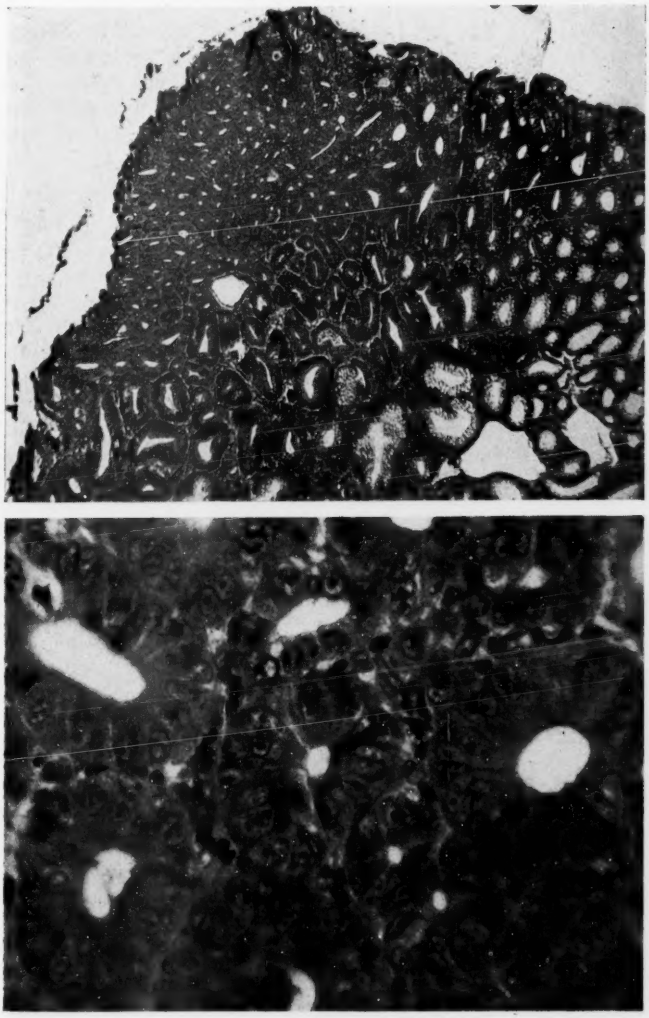
nized: (1) carcinoma in situ in which there is anaplasia and irregularity of the glandular epithelium without disruption of the basement membrane or invasion of the lamina propria (figs. 1a and b); (2) superficial carcinoma char-

(a)



(b)

FIG. 1. (a) Low power of focus of carcinoma in situ arising at edge of polyp (x50). (b) High power from same area as in 1a. Note atypical cells, macronucleoli, atypical mitoses (x450).

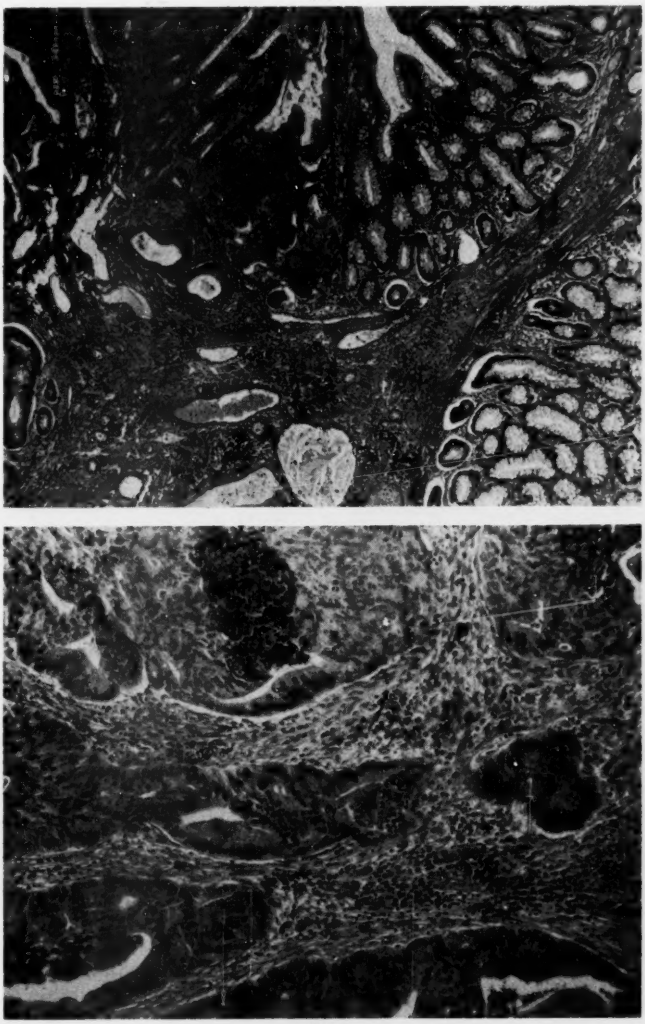
acterized by the changes mentioned previously as well as noticeable reduplication of lumina and the presence of tumor masses lying free in the lamina propria but without invasion of the muscularis mucosa (fig. 2); (3) invasive carcinoma which reveals, in addition, invasion of the muscularis mucosa with access of malignant cells to the lymphatics and vessels of the submucosa of the bowel (fig. 3a). Carcinomas without evidence of benign polyp in their structure are classified as polypoid carcinoma and do not appear in this classification. The differentiation of superficial or noninvasive carcinoma from the invasive type is, at times, difficult since the demonstration of invasion of the muscularis mucosa may be overlooked or misinterpreted because of incomplete sectioning or poor orientation of the sections or because the muscularis mucosa does not appear in the biopsy specimen. The decision for conservative or radical surgery rests upon this differentiation.

A helpful histologic aid in establishing the presence of invasion has been a desmoplastic tumor stroma associated with the invasive carcinoma arising in polyp (fig. 3b). Pathologists have long been familiar with the stromal reaction associated with epithelial neoplasms, especially of intestinal origin. However, no mention of this stromal feature in invasive carcinoma arising in polyp has been found in the literature. In reviewing 35 instances of carcinoma, the source of which is in polyp, it was noted that those tumors demonstrating invasion of the muscularis mucosa were accompanied by a desmoplastic tumor stroma. On the other hand, neoplasms classified as carcinoma in situ or superficial carcinoma arising in polyp failed to demonstrate such a stromal change.

FIG. 2. Superficial carcinoma arising in polyp at upper left. Notice glandular budding. No evidence of desmoplastic tumor stroma (x70).

Although hemorrhage and reactive changes may produce a rather dense stroma in polyp, the presence of abundant inflammatory cells and hemosiderin as well as the absence of tumor cells are helpful in the differentiation of this

(a)



(b)

FIG. 3. (a) Carcinoma invading muscularis mucosa at left (x70). (b) Another area from same tumor as in 3a with desmoplastic tumor stroma (x70).

stroma from that of invasive carcinoma. Occasionally, an inflammatory infiltrate as well as hemosiderin may be present in the desmoplastic carcinoma stroma, however tumor cells will be apparent in adjoining stromal elements.

It is admitted that the frozen section diagnosis of carcinoma arising in polyp may be difficult and somewhat inaccurate, due to technical difficulties and comparatively poor staining differential. Nevertheless, this procedure is occasionally resorted to in the diagnosis of polyps located 5 inches above the dentate line. Polypoid and sessile lesions are oftentimes removed by colonic resection but because of complicating disease, old age, or senility, a minimal surgical procedure is occasionally desired. Using the frozen section technic described by Hazard and Stevenson,[2] we have found the results satisfactory in establishing immediate diagnosis at the time of colotomy. The polyp is oriented for the pathologist by the surgeon at the operating table; it is then sectioned through the stalk into two portions with a long, sharp razor blade. One of these is used for frozen section and the other processed for permanent study. For frozen section, one portion is fixed in toto on bibulous paper by bringing the fixing fluid (alcohol 70 per cent, formalin 27 per cent, acetic acid 3 per cent) to its boiling point, and then sectioned into thin slices and once again placed in the fixing fluid and re-heated so as to insure adequate fixation. The tissue block, which has a tendency to curl, may be flattened on the freezer head by applying pressure with a glass slide during the freezing process. By this routine, the difficulties of orientation and adequate fixation and sectioning are minimized. Although cellular detail is less distinct than in permanent sections, the detection of a desmoplastic stroma associated with suspicious glandular elements has proved invaluable in the frozen section diagnosis in such lesions and has been helpful to the surgeon in his method of treatment. One of the disadvantages of such a procedure is that all areas of the polyp are not examined immediately. If the diagnosis is equivocal, the previously fixed portion, as well as the portion of tissue submitted for frozen section diagnosis, is available for permanent study. Only rarely has the frozen section diagnosis been equivocal, and under this condition the surgeon must rely upon his clinical judgment as to the most effective procedure. We favor resection under most circumstances where there is doubt.

The surgeon's cooperation with the pathologist is essential for the successful histologic study of carcinoma arising in polyp. The surgeon must supply adequate, well oriented tissue for histologic study. The entire lesion is preferable for examination, although when this is not feasible (in instances of high rectal lesions) multiple fragments from all areas have proved satisfactory.

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# TREATMENT OF ACUTE LYMPHATIC LEUKEMIA WITH AMINOPTERIN

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THERE are no specific measures for the treatment of acute lymphatic leukemia. A small number of patients experience periods of spontaneous improvement which last for varying lengths of time.[1] The most recent addition to the therapeutic agents employed for the disease has been the folic acid antagonists. Of these preparations aminopterin* has given the most promising results. The present report summarizes the response observed to aminopterin in 12 consecutive cases of acute lymphatic leukemia.

Aminopterin exerts a toxic action not only upon the granulocytic cells of the bone marrow, but also upon the erythrocytic and megakaryocytic constituents. Its effects in any given patient cannot be predicted beforehand. In acute lymphatic leukemia with lymphocytosis, aminopterin usually causes a sharp fall in the total white blood cell count but when there is lymphopenia instead of lymphocytosis the leukocyte count may either fall still further or rise toward normal. Toxic effects include pronounced hypoplasia of granulocytic and erythrocytic precursors in the bone marrow, erythroid maturation arrest at the megaloblast stage, and further suppression of platelet maturation.

The results of treatment with aminopterin are summarized in table 1 and are similar to the results reported by others.[2,3,4,5] In 5 of the 12 patients there was no detectable clinical benefit and no improvement in the blood picture. In the remaining 7 patients, however, both subjective and objective improvement occurred. In these 7 persons there was a decrease in the total number and the percentage incidence of circulating "blast cells," and in most of the patients the systemic symptoms also improved. Unfortunately, in all but one instance the improvement was temporary and persisted only for a few days to 3 months.

All patients who could be traced have since died. Four died within 3 months; 3 within 3 to 6 months; 2 within 6 to 12 months. The most favorable case is summarized in the first of the following case reports.

## Case Reports

**Case 1.** A 28 year old white man was first seen on November 5, 1950, complaining of fever, multiple joint pains and sore throat of 2 weeks' duration. There was a history of acute rheumatic fever at the age of 20 years. Physical examination revealed fever spiking to 101 F., undernourishment, and tenderness of several joints. The heart was slightly enlarged and murmurs indicative of aortic insufficiency and mitral insufficiency and stenosis were present. There were several elevated and tender erythematous lesions

*Kindly supplied by Lederle Laboratories, Pearl River, N. Y.*

Table 1

| Case | Sex | Age | Type of Leukemia | Initial W. B. C. | Initial Hgb. (Gm.) | Differential | Total Dosage Aminopterin (mg.) | Other Treatment | Complications | Results |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 28 | Acute Lymphatic | 96,000 | 10.5 | 95% Blasts. | 275 | 2500 cc. Blood | Alopecia | See Case 1 |
| 2 | M | 15 | Acute Lymphatic | 1,150 | 6.8* | 98% Lymphs; Occ. Blasts. | 17 | 4000 cc. Blood; Cortisone | Oral Ulcers | See Case 2 |
| 3 | F | 47 | Acute Lymphatic | 3,100 | 6.3* | 40% Lymphs; 27% Blasts. | 87 | 1500 cc. Blood | None | Death, 56 days<br>Subjective Improvement |
| 4 | M | 17 | Acute Lymphatic | 2,400 | 11.5* | 79% Lymphs; 6% Blasts. | 10 | 2500 cc. Blood | Oral Ulcers | Death,<br>Without Improvement |
| 5 | F | 18 | Acute Lymphatic | 4,850 | 8.5* | 93% Lymphs. | 6 | Blood | None | Death,<br>Without Improvement |
| 6 | M | 3 | Acute Lymphatic | 3,750 | 9.8 | 89% Lymphs; Occ. Blasts. | 4 | 400 cc. Blood | None | Death,<br>Without Improvement |
| 7 | M | 65 | Acute Lymphatic | 145,000 | 6.0* | 55% Blasts. | 13 | 3500 cc. Blood | Epistaxis | Fall in W. B. C.<br>Present Status not Known |
| 8 | F | 19 | Acute Lymphatic | 8,550 | 6.3* | 56% Lymphs; 27% Blasts. | 7.5 | Blood | Oral Ulcers | Subjective Improvement;<br>Present Status not Known |
| 9 | F | 60 | Acute Lymphatic | 1,650 | 6.7* | 26% Blasts. | Approx. 100 | 1500 cc. Blood | None | Death, 8 months<br>Subjective Improvement |
| 10 | F | 58 | Acute Lymphatic | 8,350 | 9.0 | 79% Blasts. | 29 | 1500 cc. Blood | Nausea; Stomatitis | Normal W. B. C.<br>Until just before Death |
| 11 | M | 4 | Acute Lymphatic | 8,950 | 13.0* | 67% Blasts. | Approx. 150 | Blood | None | Death, 4 months<br>Subjective Improvement |
| 12 | M | 9 | Acute Lymphatic | 39,500 | 7.3* | 32% Blasts. | Approx. 75 | 600 cc. Blood | None | Death, 3 months<br>Temporary Improvement |

*Transfusions prior to admission.

measuring about 1 cm. in diameter on the skin of the arms and legs. Neither lymphadenopathy nor splenomegaly was present. An initial diagnosis of recurrent acute rheumatic fever and erythema nodosa was made. Hematologic studies, however, revealed the presence of acute lymphatic leukemia. Sternal marrow inspiration disclosed a preponderance of lymphoblasts. After admission to the hospital, 1 mg. of aminopterin was administered orally each day. The hematologic response to the drug is shown in figure 1. Subjective improvement consisting chiefly of decrease of joint pains and improvement in strength began on the fifth day of treatment. The patient was discharged after 14 days, and 1 mg. of aminopterin daily was continued at home. Twenty-five days later he was readmitted for extraction of several infected teeth, and no complications occurred. Aminopterin was reduced to 0.5 mg. daily after the first month of treatment. At the end of 3 months' treatment the patient developed a mild diffuse alopecia which improved in spite of continued use of the drug.

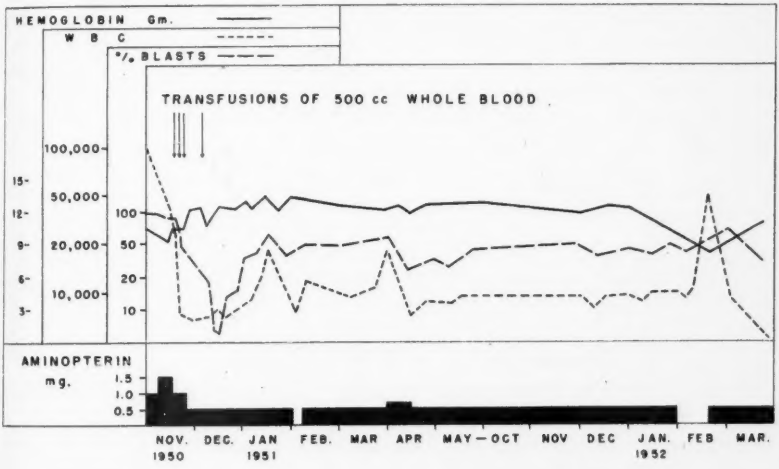


Fig. 1

Fourteen months after continuous aminopterin therapy the patient developed stomatitis, proctitis, and severe alopecia. These toxic effects subsided in 2 weeks after discontinuing the drug. The leukocyte count rose to 94,000 with 87 per cent lymphocytes and the hemoglobin fell to 9 Gm. per hundred cubic centimeters of blood. The resumption of aminopterin therapy (0.5 mg. daily) caused a prompt decrease of the leukocyte count without recurrence of the toxic effects.

The patient has been observed at intervals of 2 or 3 weeks and has worked regularly about 6 hours daily. Repeated blood smears show 10 to 20 per cent lymphoblasts, and marrow aspirations reveal no significant change in the lymphoblastic infiltration.

The course of this patient had been unusual in several respects. At no time has splenic enlargement been demonstrated, and the hemoglobin level has fallen below 11 Gm. only once in the 14 month period of observation. No blood transfusions were given except in the first 6 weeks of treatment.

The more common type of response to aminopterin is illustrated in the following case report (fig. 2).

**Case 2.** A 16 year old boy was first observed on November 18, 1950 complaining of weakness and fatigue of 18 months' duration. Bone marrow and peripheral blood studies demonstrated acute leukopenic lymphatic leukemia with severe anemia. Treatment with 1.0 mg. of aminopterin orally each day was started 3 days after admission. The fever subsided promptly but the blood picture remained unchanged. He was discharged after 10 days but was readmitted 4 days later because of gingival bleeding. The white blood cell count was 250 per cu. mm. and no platelets were seen. Aminopterin was stopped and antibiotics and transfusions of fresh whole blood were given. The patient's condition deteriorated rapidly in spite of these measures and he became semicomatose with a temperature of 105 F. The parenteral administration of 100 mg. of cortisone every 6 hours resulted in dramatic clinical improvement. The temperature fell to normal. The leukocyte count rose to 2500 and platelets reappeared in the blood smears within 36 hours. The leukocytes reached normal levels on the fifth day of cortisone therapy. Transfusions of whole blood, formerly of little benefit in raising the hemoglobin level, returned the hemoglobin to 11 Gm. per hundred cubic centimeters of blood. The patient continued a maintenance dose of 150 mg. of cortisone daily upon discharge from the hospital and improvement persisted for 6 months. At the end of this time he suddenly became worse and died. The details of the final days of his illness could not be obtained.

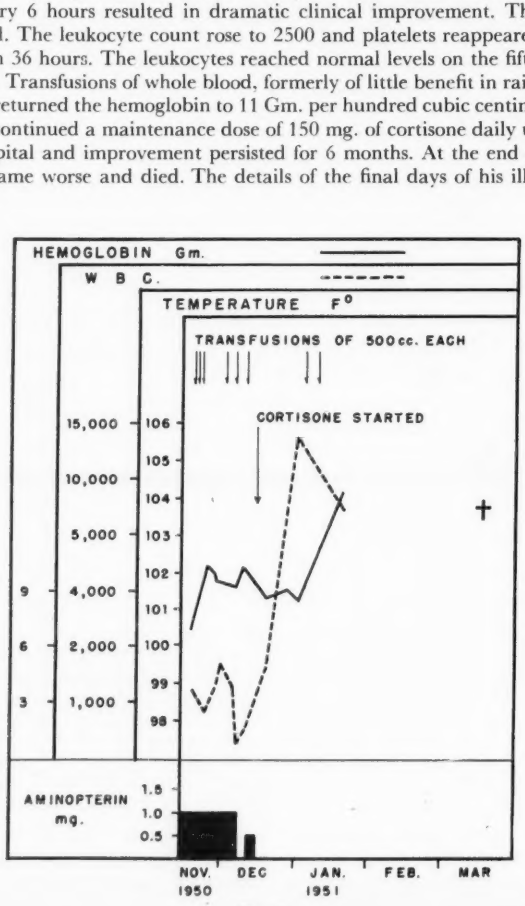


Fig. 2

## Discussion

No final conclusions regarding the benefit of aminopterin in acute lymphatic leukemia can be drawn from such a small number of patients. The response observed in the 12 consecutive patients is comparable to the reported results in large series of patients.[6] The experience of most investigators indicates that total improvement which includes complete remission, partial remission, or clinical improvement, occurs in about 50 per cent of patients treated with the drug for a minimum of 3 weeks. The fact that a reversal effect can be obtained in acute leukemia, which was formerly classified as a completely irreversible disorder, offers encouragement for future research.

## Summary

1. The effects of aminopterin in 12 consecutive cases of acute lymphatic leukemia are summarized.

2. Six of the patients derived temporary benefit from its use, the improvement lasting from a few days to 3 months.

3. One patient obtained a partial remission which persisted for 14 months after aminopterin therapy was started.

4. Five patients received no benefit from the use of the drug.

5. The striking, but temporary, beneficial effects of cortisone in a patient who had received no help from aminopterin are briefly presented.

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# BARTHOLIN'S CYSTS

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NUMEROUS therapies have been suggested for acute and chronic Bartholin's cysts. Their multiplicity indicates that no wholly satisfactory treatment has yet been found. Generally incision and drainage should be carried out in the acute stages; definite therapy instituted in the past in the chronic stages has involved destruction of the gland.

Mathieu[1] has advocated cruciate incisions with gauze wicking to assure adequate drainage. This is generally followed by excision of the cyst as described by Catell.[2] Schauffler[3] advocated cauterization of the lining wall of the cyst in both the acute and chronic stages. This method not only destroys the gland but also necessitates hospitalization.

Davies[4] attempted to overcome the disadvantages of the above procedures by incising the cysts under local anesthesia and repeatedly packing them with iodoform gauze until the drainage tract had become epithelialized. This provides a permanent new ostium and allows normal function to return.

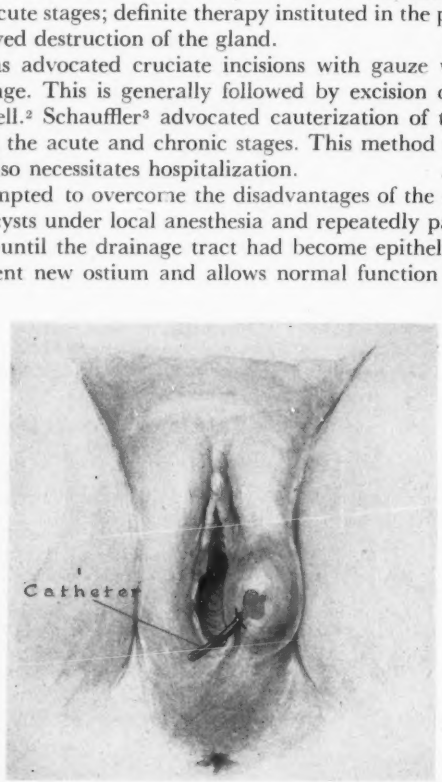


FIG. 1. Catheter drainage of Bartholin's cyst.

For the past several years, we have employed a modification of the above treatment. This was suggested by the catheter drainage of pilonidal cysts previously reported by Crile.[5] Following incision of the cyst, under local anesthesia in the office, a small mushroom catheter is inserted into the cavity (fig. 1). It is cut off close to the skin and a small safety pin is used to prevent its retraction into the cyst. The catheter provides excellent drainage and is nonirritating. It is left in place until epithelialization is complete; this is generally accomplished in 2 to 4 weeks. Patients generally become unaware of the catheter. The only complication we have seen is inadvertent removal of the tube before the tract is well established, with resultant closure. If this occurs, one need only await recurrence of the cyst and repeat the process of catheter drainage. A considerable advantage of this treatment is its application in both the acute and chronic phases of the disease.

A simplified office procedure for the treatment of Bartholin's abscesses and cysts has been presented. This therapy eliminates the expense and inconvenience of hospitalization with secondary incapacity, excludes the possibility of a frequently sanguineous operative procedure, offers primary definitive therapy, and at the same time preserves the function of a physiologically desirable gland.

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# THE SPLIT SKIN GRAFT

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EIGHTY-THREE years have passed since Reverdin described the first practical approach to the autogenous free grafting of human skin. He proposed the use of small thin bits of skin later known as "pinch grafts." Ollier and Thiersch, in 1872 and 1874 respectively, grafted larger pieces of very thin split skin, thus enlarging somewhat the scope of the graft's usefulness. There is little in the literature to suggest that skin grafts were especially popular from a surgical standpoint during the next 50 years. However, in 1929, Blair and Brown[1] introduced the thick split graft, and demonstrated vast possibilities for its use. Since then, the split skin graft has become increasingly valuable, not only to the plastic surgeon, but to the general surgeon as well. Developments in recent years have consisted mainly of ingenious machines to cut skin of uniform thickness, and at the same time eliminate much of the need for skill required in free-hand cutting of skin. The most notable of these is the Padgett dermatome,[2] which was first used in 1938 and has remained to date as the most generally useful of these instruments.

There has been a tendency in some circles to surround the grafting of skin with considerable mystery, and to suggest that grafts will "take" only for specially endowed surgeons. At the same time, surgeons are often perfectly satisfied with the survival of half or two-thirds of a graft. Both of these attitudes are regrettable. A skin graft of suitable thickness, placed on a properly prepared bed and held there by means of an adequate dressing, should survive in its entirety. Partial or complete failure of "take" is almost always due to surgical error.

## Technic

The skin is an organ of characteristic structure, consisting of an actively proliferating epidermis lying superficial to a tough, fibrous layer known as the dermis. The variation of skin thickness in different areas of the body is due mainly to the amount of dermis present. Actual measurements of skin thickness are misleading since individual variation is great. The essential point to be kept in mind by the surgeon cutting a skin graft is that he is limited as to the thickness of the graft he can take by the skin thickness of his donor site. The most common donor sites are lateral and posterior thigh, and buttock, where the skin is only slightly thinner than that of the back. Next in thickness are the skin of chest and abdominal wall, and finally the medial aspect of the thigh. When the necessity arises, there is, of course, no area which cannot be used as a donor site.

The question is frequently asked as to whether a thick or thin graft should be used. This is best answered as follows. It is generally true that the thinner

the graft, the better the chance of perfect take, particularly in the presence of contamination or poor vascularity. Conversely, the thicker the graft, the better the ultimate result in terms of minimum contracture, skin flexibility, minimum pigmentation, and durability. Therefore the thick graft, if it will take, is the graft of choice. Needless to say, if in doubt, one should err on the thin side.

### 1. Preparation of the recipient site

The clean, relatively aseptic bed produced by removing a scar or tumor, or following an avulsive injury, is ready for application of the graft. Hemostasis must be perfect; a weak adrenalin sponge may be useful in drying up annoying capillary oozing. The bed must be well vascularized, without scar, cortical bone or other material which will not nourish the graft.

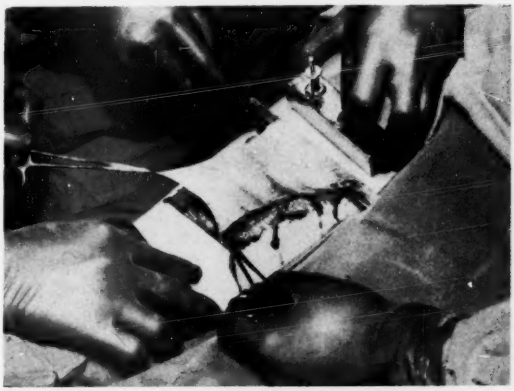


FIG. 1. In actual practice the graft is allowed to lie free; it is stretched above to show its relationship to the donor site.

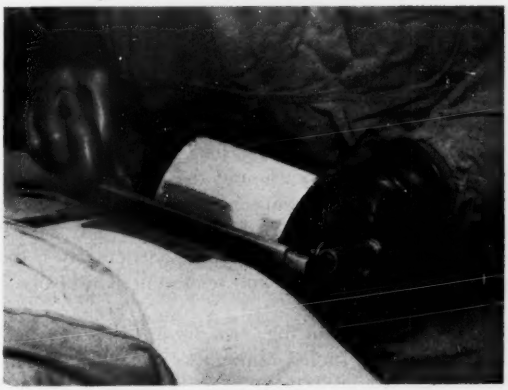


FIG. 2. The drum is rotated upward to maintain correct cutting plane.

(a) (b)

FIG. 3. (a) Preoperative view. (b) Postoperative view.

The dirty wound requires considerable preparation. Infection is combated by antibiotics administered parenterally or orally. The local wound is debrided frequently and dressed at least daily with saline dressings over nongreased fine-mesh gauze. Moderate pressure over mechanics' waste or other padding will flatten granulations and reduce local edema. Grafting should not be carried out until drainage is minimum, necrotic tissue is absent and granulations are pink and flat. These granulations may be left untouched or may be shaved off with a sharp knife just prior to application of the graft. In the case of a chronic ulcer with underlying fibrosis, the debridement is completed in the operating room with removal of all avascular tissue.

It should not have to be mentioned that the patient's general health and nutritional state are most important in obtaining a good result. These are carefully investigated at the onset of treatment. Any deficiencies in either are corrected.

**2. Cutting the graft**

While many instruments are available for cutting a split skin graft, the two most common are the free-hand knife and the Padgett dermatome. The free-hand knife has the advantages of being inexpensive, readily available, mechanically foolproof, and permitting the rapid cutting of skin. However, it is difficult to cut a perfectly uniform graft by hand, particularly if it must be thick. The dermatome, which is calibrated and set to cut skin of precise dimensions, eliminates this disadvantage.

For satisfactory free-hand cutting, two requirements must be met. First, the skin must be held taut in such a manner as to present a firm surface to the knife edge, and, second, the knife must be razor-sharp. The suction box shown in figure 1 provides an excellent means of meeting the first of these, although

(a) (b)

FIG. 4. (a) Preoperative view. (b) Postoperative view.

(a) (b)

FIG. 5. (a) Preoperative view. (b) Postoperative view.

a straight edge with counter traction by a second straight edge will do almost as well. The Ferris Smith knife with inexpensive replaceable blades is a satisfactory answer to the second requirement. These blades can be sharpened, but should be discarded if the cutting edge is not perfect. It will be immediately obvious to the surgeon taking the graft if the blade is not razor-sharp. No one can cut a good graft with a dull knife.

The skin is greased with a thin layer of vaseline to provide a seal between skin and suction box. The knife is held flat against the skin half an inch or so

away from the box, and moved in a constant plane, following the box. A little practice makes this a simple maneuver. The thickness of the graft is obvious as it is being cut; its size and shape are limited only by the donor site and the skill of the operator. As the graft is cut, the portion already free should be allowed to lie loosely behind the knife, or supported with forceps only enough to keep the cutting edge visible. Any tension at this point will result in the loss of the correct cutting plane.

When using the dermatome, a good knife blade is equally necessary. The arm is adjusted to the desired thickness, usually .015 to .025 inch, drum and donor site are cleansed with ether, and both are rapidly painted with a thin coat of dermatome cement. This cement must be thin; if too thick, it may be thinned with ether. After allowing the cement to dry for several minutes, the leading edge of the drum is held firmly against the skin for thirty seconds. The graft is then cut as the drum is rolled upward (fig. 2). Here again some practice will be necessary to cut a full drum of skin. The skin is grasped at its corners with hemostats and stripped from the drum. If cement remains on the skin, a little Biosorb will destroy its stickiness.

The graft may be kept in a sponge moistened with saline until ready for use.

**3. Application of the graft**

The graft is laid on the prepared bed without tension, and spread into position by gentle sweeps of the smooth handle of a thumb forceps. Interrupted or continuous fine silk is used to fix the graft to surrounding skin; some overlap of the graft beyond the recipient bed edge helps keep the graft in proper position. It is not necessary to "pie crust" the graft, that is, provide holes through which blood will presumably escape. The value of this procedure is strictly theoretical, and contributes nothing to a successful take. Much more important is frequent checking to be certain that flawless hemostasis is maintained.

A meticulously applied dressing will provide immobilization and maintain perfect contact between the graft and its bed. Fine mesh vaseline gauze is placed on the graft and covered with voluminous padding of mechanics' waste, fluffed gauze or abdominal pads. Moderate pressure is established by elastic or plain bandage, or Elastoplast if a circular bandage is not applicable. Immobilization and elevation of a grafted extremity are essential until the graft is well vascularized in its new bed.

The donor site is similarly dressed with considerable pressure. If left untouched, it will usually be completely epithelialized from the proliferation of hair follicle and gland epithelium within 2 weeks after operation.

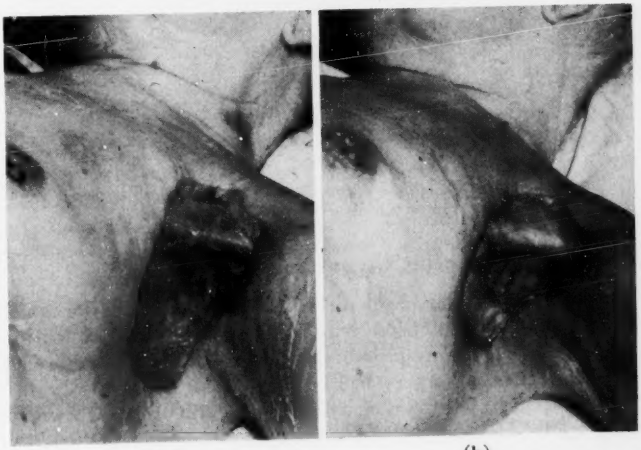
**4. Postoperative care**

Antibiotics are administered prophylactically, at least until the first postoperative dressing. This is carried out on the third or fourth day if the grafted wound was dirty, or the fourth or fifth day if it was clean. Superficial blebs containing serum are opened, and small collections of blood, if any, beneath the graft are evacuated through small incisions. The graft is trimmed as necessary, cleansed gently with soap and water, and redressed with moderate

(a)

(b)

FIG. 6. (a) Preoperative view. (b) Postoperative view.



(a) (b)

FIG. 7. (a) Preoperative view. (b) Postoperative view.

pressure. Meticulous care at this stage is just as essential as at the time of grafting.

Further dressings are done every other day or daily, with removal of sutures on the sixth or seventh day. By the tenth day the dressing usually can be reduced in size, and at 2 weeks need be no more than protection from accidental trauma. Once the dressing is off, daily soap and water cleansing, and a little lanolin or vaseline on the graft will be sufficient. If the grafted area is over or near a joint, motion is started on or about the tenth day unless the graft's survival is still in doubt.

(a)

(b)

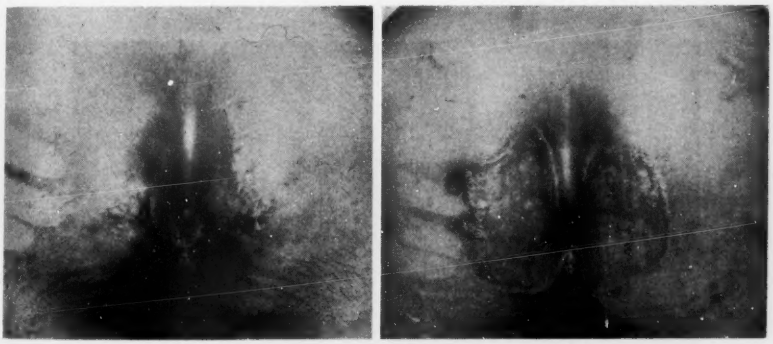
FIG. 8. (a) Shows incisions through which varicose veins had been previously excised, above and below the ulcer. (b) Postoperative view.

## Specific Uses of Split Skin Grafts

The split graft may be used for the covering of any defect for which a protective pad of subcutaneous tissue is not essential. The sole of the foot, areas with exposed tendons or bony prominences which cannot be removed, fracture sites in which additional surgery will be carried out beneath a graft, and the like, are best repaired with pedicled skin flaps carrying their own soft tissue and blood supply. However, if the surgical situation is such that immediate coverage of a defect is necessary, even these areas may be grafted with split skin as a temporary expedient.

Applications of the split graft fall roughly into two general classifications: (1) The covering of large areas denuded of skin, in which the problem is the saving of life or prevention of major deformity rather than cosmetic; (2) the repair of defects, usually operative following removal of a scar or tumor, or post-traumatic, for which primary closure is either impossible or impractical.

In the first of these categories lie burns and massive avulsive injuries such as those seen in the condition known as "wringer arm."[3] In these, while it might be preferable to provide better soft tissue replacement or to use full-thickness skin in potentially traumatized areas, the necessity of obtaining rapid coverage of raw surfaces takes precedence over functional and cosmetic considerations. If necessary, split grafts may be replaced by pedicled or full-thickness skin at a later date when the survival of the patient is no longer the primary problem. In the case of a third degree burn, grafting with split skin cannot be carried out until a satisfactory granulating bed is available, usually



(a) (b)

FIG. 9. (a) Initial lesion was pruritus ani for which considerable radiation therapy was given. Note prints in the skin of patient's fingers used to retract buttocks. (b) Postoperative view.

3 to 4 weeks after the initial injury. Grafts may be applied immediately to the healthy recipient site produced by avulsive trauma or surgery.

A typical example of this use of the graft is seen in figure 3. This child had an extensive cavernous hemangioma, rapidly approaching a size incompatible with life. Following excision, the defect which extended from axilla to groin and midline to midline was completely covered with split skin. It is interesting to note that this child's general condition, which was critical at the time of surgery, improved spectacularly as soon as skin replacement was carried out.

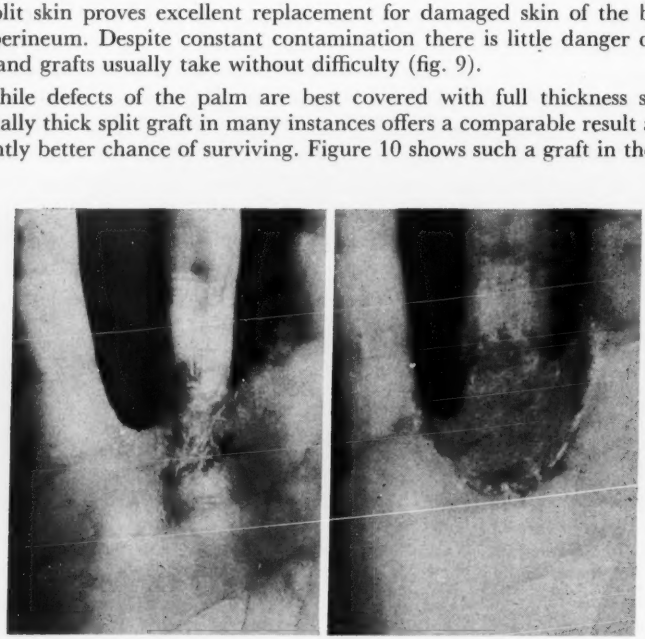
In the second group of uses of the split graft are found those instances in which the graft is the permanent covering material of choice. Figures 4 and 5 demonstrate the replacement of areas of radiation damage on the neck and face by split skin. In each case the results from both functional and cosmetic standpoints are considered satisfactory. Figure 6 shows an ulcerating carcinoma in a chronically irritated burn. A thin graft was placed on a rather poorly

vascularized base following radical excision of the lesion. The graft took well and has provided adequate coverage. In figure 7 is shown the defect remaining after radical excision of a tuberculous ulcer and associated deep scar. The function of the extremity returned to normal following skin graft and vigorous physiotherapy.

The treatment by split grafting of a typical postphlebitic leg ulcer is shown in figure 8. In this instance, the graft was laid directly on cancellous bone after removal of sclerotic cortex.

Split skin proves excellent replacement for damaged skin of the buttocks and perineum. Despite constant contamination there is little danger of infection, and grafts usually take without difficulty (fig. 9).

While defects of the palm are best covered with full thickness skin, an unusually thick split graft in many instances offers a comparable result and has a slightly better chance of surviving. Figure 10 shows such a graft in the palm:



(a) (b)

FIG. 10. (a) Preoperative view. (b) Postoperative view.

6 months postoperatively, the graft is soft and freely movable and does not interfere with the function of underlying structures.

Among less common applications of the split graft are its uses as lining for a buccal sulcus,[4] covering for the serosal surface of the bowel in the construction of an ileostomy, and as replacement for lost mucous membrane of the orbit.

In brief, a split skin graft can be applied wherever its proposed bed has sufficient vascularity to provide nourishment for the graft. Specific functional and cosmetic considerations are the only limitations to its use.

## Summary

1. The technics of cutting, applying and caring for the split skin graft have been described.

2. A brief survey of its various applications has been presented.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# ABDOMINAL TUMOR AND PROSTATIC CANCER

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UNEXPLAINED abdominal masses are seldom caused by prostatic carcinoma. Recently we observed 2 patients with large abdominal masses secondary to asymptomatic carcinoma of the prostate gland. These 2 patients emphasize the importance of a careful examination of the prostate gland in any man with an abdominal mass.

Prostatic cancer metastasizes by a number of different routes, often widely and without any symptoms from the primary tumor. The etiology of an abdominal tumor in a man may thus be overlooked. The following 2 case reports illustrate this point and demonstrate the response of prostatic cancer to antiandrogenic therapy.

**Case 1.** A man, aged 58, was first observed at the Cleveland Clinic on September 4, 1951, with a complaint of back pain of 7 months' duration. The pain, initially experienced after a fall, had not responded to any form of therapy. He had no gastrointestinal or other complaints. The patient had received treatment for syphilis several years previously.

Physical examination showed a blood pressure of 100/70; his temperature was 98 F. and pulse 60. The pertinent physical findings were limited to the abdomen and genitourinary tract. The abdomen was scaphoid, and there were no areas of tenderness. In the left upper quadrant a hard, rounded nodular mass was palpable and did not move with respiration. A similar but larger mass was also palpable in the right lower quadrant, and apparently attached to the wall of the pelvis. The prostate gland was enlarged, hard and nodular; above and lateral to the prostate were broad, hard cords of tissue.

The Wassermann test was negative; Kahn 4 plus. Blood urea was 21 mg. per hundred cc.; hemoglobin 12 Gm. per hundred cc. White blood count was 7150. Acid phosphatase was 76.5 Bodansky units; alkaline phosphatase 2.9 Bodansky units; urine pH 5.5, specific gravity 1,024, red blood cells 5-10, white blood cells 10-12 per high power field.

Roentgenograms of the spine, pelvis, and gastrointestinal tract were all essentially normal. The apices of both lung fields showed fibrocalcific infiltration, believed to represent arrested tuberculosis.

Silverman needle biopsy of the prostate gland revealed adenocarcinoma with intravascular and perineural lymphatic extension. A diagnosis was made of carcinoma of the prostate gland with metastasis to the retroperitoneal glands. Bilateral orchiectomy was performed and estrogenic therapy begun in the form of stilbestrol (1 mg. four times daily).

Within 1 month the abdominal masses had regressed in size and become softer. The prostate gland likewise had become smaller and softer. The acid phosphatase was 6.1 and the alkaline phosphatase 1.7 Bodansky units. Four months later the abdominal masses were no longer palpable. The prostate gland was soft, but there was still evidence of extension of the carcinoma in the region of the seminal vesicles. Acid phosphatase at this time was 1.6 Bodansky units.

**Case 2.** A man, aged 58, came to the Clinic with the chief complaint of a mass in the rectum. For 7 to 8 months prior to observation he had experienced progressive constipation which was relieved by cathartics several times a week. For 2 months he had noticed blood in the stools, had lost his appetite and 10 pounds in weight. Three weeks previously he had experienced an episode of urinary frequency, dysuria, urgency and nocturia.

On physical examination blood pressure was 160/92 and temperature 99.4 F. Heart and chest were essentially normal. On examination of the abdomen, the liver edge was just palpable. In both the right upper and left lower quadrants there were hard, smooth, rounded masses. The former was movable and somewhat tender; the latter mass was less tender. Rectal examination showed a large mass in the region of the prostate gland which extended up on the right side of the pelvic wall and underneath the base of the bladder. It was hard, nodular and irregular.

Hemoglobin was 12 Gm., and white blood count 9400. Serum phosphorus was 3.2; acid phosphatase 8.5 Bodansky units. Blood sugar was 81 mg. and blood urea 24 mg. Roentgenograms of chest and KUB plates of the abdomen were essentially normal.

Needle biopsy of the rectal mass was performed but the tissue was not adequate for diagnosis. The proctoscopist suspected Blumer's shelf and an inoperable carcinoma of the bowel with metastasis to the peritoneal floor. The hard nodules in the prostate and the elevated acid phosphatase suggested a carcinoma of the prostate gland.

The patient was placed on a low residue diet and was given stilbestrol, 1 mg., 3 times a day. He returned 4 months later complaining of attacks of right lower quadrant pain which had continued from 3 to 4 days. Physical examination revealed that the hard masses in the abdomen had disappeared. The prostate was again found to be enlarged and hard with extension of the induration laterally. Gastrointestinal and genitourinary roentgen studies were normal except for nonvisualization of the gallbladder after 2 doses of dye.

Three months later the patient had several attacks of severe epigastric pain associated with vomiting. This was thought to be cholecystitis. Acid phosphatase at this time was 0.9 Bodansky units and alkaline phosphatase 4.4 Bodansky units. Blood urea was 21 mg. and hemoglobin 11.5 Gm. The patient was advised to continue stilbestrol, 1 mg., 4 times a day. The last visit was on February 14, 1952 (12 months after the original visit), at which time he was asymptomatic. Examination of the abdomen was negative; the prostate showed hardness of the right lobe with lateral extension to the wall of the pelvis.

## Discussion

Palpable retroperitoneal iliac or aortic lymph nodes are found in a small number of all men with prostatic cancer.[1] This does not reflect the actual involvement of these lymph nodes by this neoplasm because, in most instances, the glands are not large enough to be felt through the abdominal wall.[2] Perineural lymphatics within the prostate gland are involved early by carcinoma

and probably also the capillaries of the blood stream. Metastasis to the bony pelvis and spine occurs via the lymphatics associated with the periprostatic and pelvic nerves[3] and the venous channels which lead to the bony structures.[4] The regular pelvic lymphatics are also invaded with gradual spread to the iliac and aortic glands with later extension to the entire lymphatic system.

In practically all instances where there is evidence of metastatic prostatic cancer, the prostate gland is abnormal, presenting hard nodularity with palpable evidence of extension of the process beyond the gland. Occasionally the prostate may be small and hard but the metastasis around it extensive, making the observations difficult to interpret.

Other diseases which cause retroperitoneal gland enlargement are sarcoidosis, tuberculosis, lymphoma, and metastatic carcinoma. Primary retroperitoneal tumors such as lipomas, sarcomas, or neurofibromas, must also be considered in the diagnosis. Tuberculosis and lymphoma may involve the prostate gland but the palpatory findings are not those of prostatic cancer.

The diagnosis of carcinoma of the prostate with metastasis is supported by the discovery of a high serum acid phosphatase. If the cancer is confined to the prostate gland there is no elevation of the serum acid phosphatase. Normal levels are often found when there is extensive disease, the tumor cells apparently being incapable of producing acid phosphatase. The diagnosis is also supported by roentgen evidence of osteoblastic metastasis characteristic of carcinoma of the prostate; with such osseous lesions both serum alkaline and acid phosphatase are elevated in most instances. A biopsy of the prostate gland is obtained readily by direct perineal puncture with a biopsy needle, with a resectoscope from the prostatic urethra in suitable cases, or by open perineal biopsy.

The presumptive diagnosis of prostatic cancer in the retroperitoneal glands is made if the tumor becomes smaller under appropriate antiandrogenic therapy, as with the 2 patients discussed here.

The possibility of metastasis from carcinoma of the prostate should be considered in any man with a questionable abdominal mass.

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# EOSINOPHILIC GRANULOMA, BILATERAL MASTOID INVOLVEMENT

## *A Case Report*

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IN review of the literature it was found that xanthomatous lesions of the temporal bones are not unusual, but that only 3 cases of bilateral mastoid involvement have been reported[1,2,3] since Schuller and Christian discussed, in 1915 and 1919 respectively, cases presenting a syndrome now known as Hand-Schuller-Christian's disease. Hand's name became associated with this syndrome because he reported one case in 1893 and another in 1921 which presented the clinical triad of diabetes insipidus, exophthalmos, and bony defects of the skull—the findings necessary to establish the diagnosis of Hand-Schuller-Christian's disease. He attributed this to tuberculosis, however.

In 1932 Greifenstein[4] reviewed the 26 cases of Hand-Schuller-Christian's disease appearing in the literature up to that time and noted that the ear was involved in 12 of these cases. Not until 1935 did an abundance of literature on Hand-Schuller-Christian's disease or eosinophilic granuloma appear in the journals; several years later the true relationship between these two entities and Abt-Letterer-Siwe's disease was established.

The histopathology involved in this syndrome was defined in 1928 by Rowland[5] who concluded that it consisted of a form of xanthoma involving the reticulo-endothelial system of certain areas of the skull. However, it was not until 1940 to 1948 that the close association of Abt-Letterer-Siwe's disease, Hand-Schuller-Christian's disease and eosinophilic granuloma, as variants of the same xanthomatous disease, was recognized.[6]

In view of the infrequent instances of bilateral mastoid involvement by eosinophilic granuloma found in the literature, it was felt that this case would provide an interesting report. In addition, a not unusual diagnostic error is demonstrated.

The patient, a 3 year old white girl who apparently had been in good health for the first 2 years of life, was seen in the Department of Neurosurgery of the Cleveland Clinic in September 1950. At that time a diagnosis was made of eosinophilic granuloma in the right parietal area of the skull. The roentgenologic diagnosis (fig. 1) was confirmed histologically at the time a cranioplasty was performed, the involved area removed, and the defect covered with tantalum. She was sent home and not seen again until January 11, 1951, at which time she was first observed in the Department of Otolaryngology. She had been brought into the Clinic because of a chronically draining wound behind her left ear. The history revealed that shortly following the previous hospital discharge, she developed a slight swelling behind the ear. The family physician felt that this was an infectious process as a result of the child's scratching this area, especially

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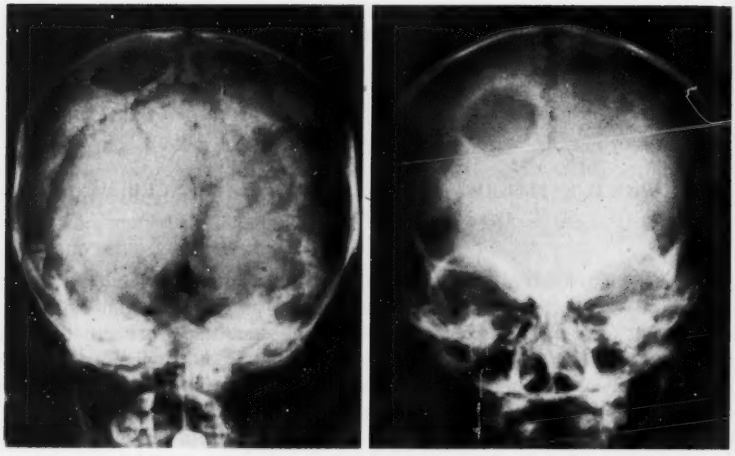

FIG. 1. Other portions of skull involved as shown by the circular area of radiolucency involving both inner and outer tables of skull in posterior parietal region.

in view of the fact that no evidence existed of otitis media and no history of otalgia. There was, however, neither fever, chills, nor pain. The mass was incised by the family physician and pus drained from the wound. The wound continued to drain serous material and showed poor healing tendencies. When seen in the Clinic, a draining fistula behind the left ear was observed. Also, considerable debris was encountered in both external auditory canals. After this debris had been removed, it was noted that there was a bulging of the posterior canal wall into the lumen of the left external auditory meatus. Mastoid x-rays were taken bilaterally which revealed almost identical destructive lesions. The x-rays were so similar that the possibility was considered an error had occurred in marking and the films were repeated with special attention that right and left films were obtained and correctly identified. The repeat films demonstrated radiolucent punched-out or destructive areas in both mastoid processes and both petrous ridges (fig. 2). This roentgenographic evidence, in addition to the previously obtained pathologic evidence at the time of the cranioplasty, would be adequate for diagnosis. However, on January 24, 1951, a biopsy was taken from the bulging area of the posterior canal wall of the left external auditory meatus. This was reported as follows:

"Pathologic diagnosis: eosinophilic granuloma (biopsy left external auditory canal).

Gross: Three fragments of tissue, averaging about 3 mm. in diameter.

Microscopic: Section reveals tiny fragments of tissue formed principally by granulation tissue containing frequent mononuclear cells with acidophilic and neutrophilic cytoplasm, nuclei oval, round or indented and occasionally with several nucleoli. Intermingled with these are cells, principally eosinophilic with segmented nuclei. Capillaries are less in evidence than in usual granulation tissue. Also present is a tissue fragment covered by well differentiated, keratinizing squamous epithelium, underlying tissue being infiltrated by many eosinophiles and a few histiocytes."

Therapy consisted of seven x-ray treatments to each temporal bone, totalling

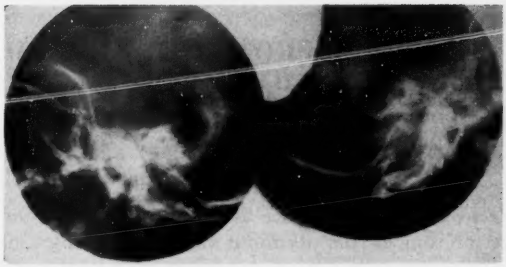

FIG. 2. Roentgenograms reveal large destructive areas in both mastoid regions resembling operative defects.

850 r each side. Approximately one week following completion of the x-ray therapy, the postauricular fistula was healed and the bulge in the left auditory canal wall had receded.

This child has been observed at numerous intervals, subsequently, and the left ear canal remains dry and the postauricular sinus tract is closed with no evidence of postauricular swelling. However, on January 10, 1952, the child returned because of a postauricular swelling and discharge from the right (opposite) ear. At that time, further x-ray therapy was given to the right mastoid region and one week later the discharge had subsided and the ear again appeared to be normal.

## Summary

A case of eosinophilic granuloma is reported with the unusual finding of bilateral mastoid involvement, as proved on biopsy.

The roentgenographic findings in the mastoid bones resemble operative defects. The associated infection and swelling of the ear canals, as well as the postauricular sinus tract, responded well to x-ray therapy.

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# BLEEDING FROM THE RECTUM

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BLEEDING from the anal orifice is a sign which demands investigation. Since the rectum is the terminus of the gastrointestinal tract, blood which emanates therefrom may represent any portion from mouth to perineum. Careful questioning of the patient regarding the character of the bleeding is important, since blood originating from any part of the tract is fairly characteristic of that section. In general, red blood represents bleeding from the cecum downward, and black bleeding (melena) from the mouth through the small intestine. However, exceptions may occur. Truly massive hemorrhage from esophageal varices, hiatus hernia, gastric neoplasm or peptic ulcer may irritate the small and large intestine so that peristalsis will hurry blood to the rectum while it is still bright red. Fortunately, vomiting which occurs in most cases of massive gastric hemorrhage will suggest localization.

## Melena

Melena, or tarry stool, represents blood altered by its passage through the small intestine. This form of blood may appear mixed with stool or as sticky, foul smelling, tarry masses. If such blood has come through the upper tract more rapidly than chyme, it may not have been completely changed and may appear dark red or impart a Burgundy wine color to the water in the bowl. Such stool gives a four plus benzidine or guaiac reaction. Iron compounds prescribed for anemia produce a black, tarry stool. Ingestion of beets produces a dark red stool, sometimes assumed to be blood. Table 1 lists the common causes of melena.

## Colonic and Rectal Bleeding

Bright red blood usually has its origin in the colon or rectum and careful evaluation of the patient's history serves to establish the source in most cases. Such localization is important. We have seen patients subjected to complete gastrointestinal investigation and exploratory laparotomy for anemia due to hemorrhoidal bleeding. A few minutes spent in determining the character of the bleeding would have saved unnecessary expense and a major operative procedure.

Blood of colonic origin is always *mixed* with stool unless the bleeding is sudden and profuse, in which case it may appear as a mass of clots. Carcinoma, polyps, and ulcerative colitis produce blood that is churned with stool and most patients will volunteer this information if questioned carefully. Large papillary lesions of the cecum or ascending colon may bleed constantly, but

in such small amounts that it escapes the patient's detection. Sudden massive hemorrhage from the colon is usually due to a benign lesion.

Benign or malignant lesions as low as the rectosigmoid most often produce blood mixed with stool but, below this point, it may be described as "on stool." Bleeding from the hemorrhoidal area and anal canal can usually be differentiated by relation to passage of stool and whether or not blood comes from inside or outside the sphincters.

Hemorrhoidal bleeding follows the passage of stool and is described by the patient as "streaks" on the stool, or blood in the bowl. More severe bleeding is in the form of "squirts" or "drips." The entire perineum may be bathed in blood. This is explained by the sudden release of the tamponade effect of the stool on the hemorrhoid at the time of greatest effort and pressure. As the turgid prolapsing hemorrhoids retract, they are stripped by contracting sphincters.

Other sources of bleeding are anal papilla and fissure in ano. These are traumatized by the passage of stool leaving a small amount of blood on the anal margin which is picked up by tissue. Chronic recurrent fissures or anal scars that are epithelialized and therefore inactive often are the seat of minute telangiectases that bleed following the passage of hard stool. This blood is also seen on the tissue and its source is not apparent on casual examination.

Blood which appears on undergarments has its source outside the sphincter. The commonest causes of this type of bleeding are permanently prolapsed hemorrhoids, or rectal mucosa and prolapsed ulcerated anal papillae. An occasional prolapsed polyp may also be responsible for bleeding of this nature.

Other causes of bleeding from the colon may be obscure and almost impossible to locate. Among these are ruptured telangiectases, submucosal infarcts, the per rhexin hemorrhage of hypertensives, the obscure bleeding from diverticulosis, and that associated with hemorrhagic diatheses.

## Diagnostic Procedures

Patients with melena as a sign of bleeding should be subjected to complete upper gastrointestinal investigation. Red blood from the rectum, however, should require initial rectal and colonic examination. Anoscopic and proctosigmoidoscopic examination, followed by colon x-rays with double air contrast technic are indicated for those exhibiting blood mixed with stool. If bleeding is of the anorectal type, i. e., follows stool or "on stool," or appears on the tissue, anoscopic and proctosigmoidoscopic examination may be sufficient, provided the cause of bleeding is obvious. If there is any change in bowel habit, no matter how slight, colon x-ray is indicated to eliminate carcinoma. Double air contrast technic should always be employed where blood is mixed with stool (if barium enema is negative), since this may be the only indication of polyp.

## Self Observation

A fairly accurate diagnosis can usually be made from the patient's statements prior to examination. Detailed self-observations on the duration, amount,

and frequency of bleeding are common. There is minute accuracy on such points as the relation of bleeding to the passage of stool and whether it is mixed or not. Most patients can describe a rectal prolapse, protruding internal hemorrhoids or a prolapsed hypertrophied anal papilla. Indeed, more often than we suspect, the pathologic condition has been viewed with a mirror. It only remains for the physician to bring out the details of these valuable observations.

The appearance of red blood at the anal orifice has its origin in the majority of instances in the anorectal area and the immediate 20 centimeters above this point. Careful history taking and proctosigmoidoscopic examination will result in an accurate diagnosis in most cases. In others, careful repeated roentgen investigation may be necessary and multiple proctosigmoidoscopic examinations are of definite value.

Table 1

**Causes of Melena**

| |
|---|
| Peptic ulcer |
| Hiatus hernia |
| Esophageal varices |
| Meckel's diverticulum |
| Small bowel tumors |
| Tumors of stomach |
| Anomalies of small intestine (with ulceration) |
| Bleeding gastritis |

Table 2

**Causes of Bleeding from the Large Intestine**

| | |
|---|---|
| Hemorrhoids | |
| Carcinoma | |
| Adenomas (Polyps) | |
| Ulcerative colitis | |
| Intussusception | |
| Obscure origin | Telangiectases |
| | Diverticulosis |
| | Hemorrhagic diatheses |
| | Per rhexin hemorrhage |

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### ERRATUM

On page 39, line 6, of the January 1952 *Quarterly* substitute *submaxillary salivary gland* for *submaxillary and salivary gland.*